ring. This indicates that besides being a mechanical structure that undergoes constriction, the actomyosin ring plays an additional and important role in cytokinesis. We have reported that the conserved GTPase Cdc42 is activated in a unique spatiotemporal manner. Cdc42 activation pattern during cytokinesis depends on the localization of its activators, Gef1 and Scd1. Gef1 promotes the recruitment of the septum synthesizing enzyme Bgs1 at the ring to allow timely onset of ring constriction and septum ingression, while Scd1 is required for primary septum formation. Here we show that Gef1 has a role in promoting concentric furrow formation. Gef1 contributes to the uniform distribution of the F-BAR protein Cdc15 and Bgs1 along the ring. Cdc15 promotes recruitment of Bgs1 to the division site to enable septum ingression. Cells with uneven Cdc15 and Bgs1 distribution along the ring display abnormally long constriction times. In addition, regions of the ring with decreased Cdc15 show delayed rates of constriction resulting in non-concentric furrowing. This suggests that the Cdc42 GEF Gef1 enables the actomyosin ring to act as a landmark and guide for proper organization of proteins at the division site to ensure efficient septum ingression and furrow formation.

P1850

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Cytoskeletal crosslinkers both drive and brake cytokinetic ring closure.

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Cell shape changes in cytokinesis are brought about by a transient structure historically known as the contractile ring, rich in non-muscle myosin II (NMM II) motor proteins and actin filaments (F-actin). It has becoming increasingly appreciated that ring closure can be driven not only by myosin motor activity but rather by F-actin depolymerization coupled to crosslinking. We used the *C. elegans* zygote to study the role of F-actin crosslinkers in cytokinetic ring closure. Thorough depletion of anillin did not affect furrowing speed, but partial depletion allowed faster furrowing. Similarly, while thorough depletion of NMM II slowed ring closure, partial depletion allowed a faster maximum furrowing speed. Partial NMM II depletion also allowed faster furrow initiation. We measured the effect of tuning myosin levels on cytoskeletal organization during early cytokinesis and found partial myosin inhibition accelerates the transformation of the initially isotropic F-actin meshwork into circumferential bundles.
These results suggest that crosslinkers brake furrowing by dampening cytoskeletal bundling, alignment, and potentially sliding, in the division plane. Together with others' recently published findings of work with in vitro reconstituted and in silico rings, our findings support the conclusion that, in addition to contributing to the driving force of ring closure, crosslinkers also resist ring closure.

P1851

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Constriction mechanism of the actomyosin ring.

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Cytokinesis in most eukaryotic cells is orchestrated by a contractile actomyosin ring. While many of the proteins involved are known, the mechanism of constriction remains unclear. Based on the current

literature and new 3D molecular details from electron cryotomography, here we developed 3D coarse-grained models of actin filaments, unipolar and bipolar myosins, actin crosslinkers, and membranes and simulated their interactions. Exploring a matrix of possible actomyosin configurations showed that node-based architectures like those presently described for ring assembly result in membrane puckers not seen in EM images of real cells. Instead, actin and myosin are more likely uniformly distributed around the ring. In the model that best matches data from fluorescence microscopy, electron cryotomography, and biochemical experiments, ring tension is generated by interactions between bipolar myosins and actin, and transmitted to the membrane via unipolar myosins.

P1852

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Back-to-back mechanisms drive actomyosin ring contraction during *Drosophila* embryo cleavage.

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Contraction of actomyosin rings during cytokinesis is typically attributed to actin filaments sliding towards each other via Myosin-2 motor activity. However, ring contraction proceeds in some cells even in the absence of Myosin-2 or its motor activity. Thus, ring contraction uses both Myosin-2 dependent and independent mechanisms. But what the Myosin-2 independent mechanisms are, and to what extent they are sufficient to drive contraction on their own remains unclear. Here, we show that during the first complete cytokinesis in *Drosophila* embryos, actomyosin rings contract in two sequential and mechanistically distinct phases. The first contraction phase is Myosin-2 dependent, and the second contraction phase is Myosin-2 independent. The mechanisms driving each phase are genetically and pharmacologically separable; and Myosin-2 independent contraction alone requires F-actin turnover. Finally, our data suggests that switching from Myosin-2 dependent to independent contraction relies on the spatial organization of Factin as controlled by Cofilin, Anillin, and Septin. Cellularization now offers the opportunity to compare Myosin-2 dependent and independent contraction in the same cell type, without concern for overlap between mechanisms.

P1853

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Component turnover in the cytokinetic ring maintains organizational homeostasis and tension production.

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Why do components turn over in the cytokinetic contractile ring? Turnover of actin, myosin-II and other components is a fundamental feature of the ring, an actomyosin machine whose components are organized to generate tension and help divide the cell into two. A major obstacle is that interfering with turnover processes is experimentally challenging.